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Can inflammatory markers predict intestinal wall ischemia in sigmoid volvulus?

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ABSTRACT

Aims: Sigmoid volvulus (SV), which causes closed-loop obstruction as a result of the rotation of the sigmoid colon around its mesentery, causes serious morbidity and mortality, especially in elderly patients, when necrosis develops in the intestine. In this study, it was investigated whether inflammatory markers, which guide the diagnosis and prognosis of many intra-abdominal pathologies, can predict the presence of ischemia in the intestine in patients with SV.

Methods: In the study, patients who were operated on for SV in our clinic between January 2019 and March 2022 were retrospectively screened, and the demographic, clinicopathological, and laboratory parameters of the patients were examined. Data were analyzed with the Student's T test and Mann-Whitney U-test.

Results: Of the 47 patients included in the study, 28 (59.6%) were male and 19 (40.4%) were female. Intestinal necrosis was detected in 23 (48.9%) patients who underwent surgery. The mean age of the patient population was 70 (±19). There was no statistical difference between the two groups according to age (p=0.338). White blood cell count (WBC) (p=0.686); Neutrophil count (p=0.949); Lymphocyte count (p=0.790); Monocyte count (p=0.898); Neutrophil-lymphocyte ratio (NLR) (p=0.733); Platelet count (p=0.766); RDW value (p=0.725); There was no statistically significant difference between the two groups in lactate level (p=0.289), C-reactive protein (CRP) (p=0.212), Amylase (p=0.975) and Lactate dehydrogenase (LDH) (p=0.974) levels.

Conclusion: Detection of intestinal necrosis is a vital condition for the patient. Inflammatory biomarkers obtained from preoperative routine blood parameters were not found to be significant in estimating the presence of intestinal necrosis in the SV clinic. It was concluded that large series are needed in this regard.

Keywords: Sigmoid, volvulus, ischemia

INTRODUCTION

Colonic volvulus is the rotation of a redundant segment of the colon around its mesentery, which causes obstruction in the volvulus developing segment and proximal and may cause ischemia, necrosis, and perforation in the colon due to impaired blood flow.[1] The sigmoid volvulus (SV) was first described by von Rokitansky.[2] SV is one of the three most common causes of acute colonic obstruction and accounts for 50-90% of all colonic volvulus.[3] SV, which is more common in men; Africa, Asia, the Middle East, South American countries, and Turkey are endemic areas, and the incidence for the US population is 0.001% per year.[4]

Although intestinal vascularization and necrosis can be evaluated with Ultrasonography, angiography, and Scintigraphy, it has been reported that intestinal viability can be demonstrated by endoscopic examination.[5] Lower intestinal gastrointestinal endoscopy is recommended to evaluate sigmoid colon viability, detorsion, and decompress the colon in hemodynamically stable patients without evidence of peritonitis or perforation.[1] If necrosis or perforation has developed in the wall of the abdomen, if there are signs of generalized peritonitis in the abdomen, if there is an early recurrence after unsuccessful endoscopic detorsion or detorsion, emergency surgery is indicated in these patients.[6] Intestinal necrosis develops in 6.1-93.4% of SV cases, which is a catastrophic complication that doubles mortality.[7] In this study, it was aimed to investigate the effectiveness of inflammatory markers, which are included in the diagnostic algorithm of many intra-abdominal pathologies, in predicting intestinal wall necrosis in patients diagnosed with SV.

METHODS

Patients

This study was conducted in Ankara City Hospital between January 2019 and March 2022, after the approval

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of Ankara City Hospital No:1 Clinical Researches Ethics Committee (Date: 12.01.2022, Decision No: E1-22-2275). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The patients who were operated on for SV between the two groups were screened retrospectively, and demographic, clinico-pathological, and laboratory parameters of the patients were obtained from electronic records. The data of 47 patients older than 18 years of age who were operated on with the diagnosis of SV, whose electronic records were accessed, was analyzed. Patients who were admitted to the emergency room with the diagnosis of SV and underwent colon resection by planning an emergency surgical intervention due to the presence of acute abdominal symptoms were included in the study. Patients who did not have radiological findings of acute abdomen, generalized peritonitis, colonic perforation, or full-thickness necrosis in the colon wall at the time of admission to the emergency department, patients who underwent endoscopic detorsion, patients under the age of 18, patients with any malignancy, and patients using immunosuppressive drugs were not included in the study. Resection materials were examined by expert pathologists. Patients were divided into two groups according to the presence (Group A) or absence (Group B) of pathological necrosis in the postoperative histopathological examination of the resection material.

Materials

The age and gender of the patients, hemogram taken at the first admission to the hospital, biochemical parameters, and venous blood samples were examined, and the results of the study were obtained from these sample analyses. White blood cell count (WBC) (reference range: 440-11300/uL), neutrophil count (reference range: 110-9600 uL), lymphocyte count (reference range: 500-6000 uL), monocyte count (reference range: 100-1400 uL), RDW value (reference range: 11.5-16%), neutrophil-lymphocyte ratio (NLR), and platelet count (reference range: 150.000-500.000/uL), C-reactive protein (CRP) (reference range: 0.15-5 mg/dl), amylase (reference range: 30-118 U/L), lactate dehydrogenase (LDH) (reference range: 120-246 U/L), and venous blood gas Lactate level (reference range: 4.5-20 mmol/L) was recorded.

Statistical Analysis

Statistical Analysis Statistical analyses were performed using the IBM Statistical Package for the Social Sciences version 26 (IBM SPSS Corp.; Armonk, NY, USA). The compliance of numerical data with a normal distribution was checked with the Kolmogorov-Smirnov test. It was determined that the WBC, Lymphocyte, Monocyte, Platelet, lactate, and RDW values of the variables showed normal distributions, while the others did not show normal distribution. Among these variables, the median (quarter percent) values for the non-parametric distributions and the mean (±Standard Deviation) values for the parametric distributions were given (**Table 1**). Continuous variables were analyzed with the Student's T test and the Mann-Whitney U-test. $p<0.05$ was considered statistically significant.

RESULTS

Of the 47 patients included in the study, 28 (59.6%) were male and 19 (40.4%) were female. The mean age of the patient population was 70 (±19). The mean age of 23 patients in Group A was 73, and the mean age of 24 patients in Group B was 66. There was no statistically significant difference between the two groups according to age (p=0.530). While mortality developed in 5 (21%) patients in Group A, Mortality developed in 4 (16%) patients in Group B. WBC (p=0.714); Neutrophil count (p=0.798); Lymphocyte count (p=0.282); Monocyte count (p=0.633); Platelet count (p=0.299); RDW value (p=0.951); NLR (p=0.625); Lactate level (p=0.113), CRP (p=0.134), Amylase (p=0.774) and LDH (p=0.783) levels were not statistically significantly different between the two groups (**Table 2**).

Table 1. Clinical and demographic data of the patients

	N	
Age	47	76 (56-85)**
Neutrophil count	47	7.93 (5.6-9.6)**
NLR	47	6.87 (3.8-12.2)**
LDH	46	256 (213-309)**
Amylase	47	48 (34-75)**
CRP	32	18 (3-56)**
WBC	47	10.47 (±4.13)*
Lymphocyte count	47	1.19 (±0.58)*
Monocyte count	47	0.53 (±0.25)*
Platelet count	47	279 (±89)*
RDW	47	14.7 (±1.5)*
Lactate	30	2.01 (±1.09)*

Notes: Parametric variables are given as mean (±standard deviation) * if they are normally distributed and as median (quarter percent) ** if they are not normally distributed.
Abbreviations: NLR, neutrophil-to-lymphocyte ratio; LDH, lactate dehydrogenase; CRP, C-reactive protein; WBC, white blood cell count.

Table 2. Continuous variables and categorical data

	Group		p
Age	Group A Group B	78 (55-82)** 73 (57-85)**	0.530
Neutrophil count	Group A Group B	8.32 (5.6-9.4)** 7.78 (5.5-12.1)**	0.798
NLR	Group A Group B	6.68 (3.9-12.2)** 8.38 (3.8-12.8)**	0.625
LDH	Group A Group B	254 (214-276)** 262 (209-321)**	0.783
Amylase	Group A Group B	49 (31-94)** 47 (43-67)**	0.774
CRP	Group A Group B	35 (11-64)** 11 (3-43)**	0.134
WBC	Group A Group B	10.2 (±3.8)* 10.6 (±4.4)*	0.530
Lymphocyte count	Group A Group B	1.2 (±0.6)* 1.1 (±0.5)*	0.282
Monocyte count	Group A Group B	0.5 (±0.2)* 0.5 (±0.2)*	0.633
Platelet count	Group A Group B	292 (±86)* 265 (±92)*	0.299
RDW	Group A Group B	14.8 (±1.6)* 14.7 (±1.4)*	0.951
Lactate	Group A Group B	2.3 (±1.1)* 1.6 (±0.9)*	0.113

Notes: Parametric variables are given as mean (±standard deviation) * if they are normally distributed and as median (quarter percent) ** if they are not normally distributed.
Abbreviations: NLR, neutrophil-to-lymphocyte ratio; LDH, lactate dehydrogenase; CRP, C-reactive protein; WBC, white blood cell count.

DISCUSSION

SV is one of the leading causes of large bowel obstruction worldwide. Both luminal obstruction and vascular occlusion, which develop due to the rotation of the sigmoid colon around its mesentery, are the causes of the pathophysiological changes and clinic in the SV. Loss of volume in the obstructive intestinal lumen in the SV leads to hypovolemia as well as mucosal ischemic damage; necrosis causes toxicity

by facilitating bacterial translocation and absorption of toxic products.[8]

The first step in SV diagnosis is visualization of the diagnosis, mainly by plain radiography and computed tomography. Colonoscopy is then usually performed to further confirm the diagnosis and, if possible, to try to detorsion the SV endoscopically.[9] Colonoscopic detorsion is the first-line treatment if there is no evidence of peritonitis.[10] It is difficult to determine the extent of mucosal ischemia/necrosis during endoscopy. A completely black mucosa defines full-thickness necrosis.[11] Emergency surgery is considered inevitable in this case.[6] However, in borderline ischemia, the black mucosa contains different colors (yellow, reddish spots). In this situation, emergency surgery can be avoided with conservative treatment through close monitoring with intermittent colonoscopies (1-2 days), abdominal examinations (6-8 hours), and laboratory tests (twice a day) after detorsion.[11] When he sees signs of some ischemic changes in the sigmoid colon, he is concerned about the decision to follow the patient or have an emergency surgery.[9] In our study, in such cases, inflammatory markers were found to be ineffective in guiding clinicians and supporting patients with intestinal necrosis without acute abdomen symptoms but with intestinal necrosis without wasting time with an invasive and morbid procedure such as colonoscopic detorsion.

Sigmoid necrosis develops in 6.1% to 93.4% of SV cases.[12] While 17-100% mortality develops, in this case, the mortality rate in the absence of necrosis is 3-30%.[13] In our study, mortality was 21% in those with intestinal necrosis and 16% in those without necrosis, which was consistent with the literature.

In the current literature, intracellular enzymes, intracellular electrolytes, and inflammatory cells/markers; in addition to the increase in LDH and CRP, the reasons for the increase or decrease in the number of leukocytes are thought to be due to necrosis in the intestinal tissue and it is supported by studies. Khan et al.[14] showed that hematological markers can be helpful in considering the diagnosis of acute mesenteric ischemia in emergency situations. With this, Bostancı et al.[15] showed that preoperative elevation of NLR, Platelet-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) values in patients with incarcerated abdominal wall hernia may indicate intestinal ischemia and bowel resection ($p<0.05$). Ceylan et al.[16] In the study conducted by SV, they created a model that aims to predict whether there is intestinal necrosis in patients with SV only by blood tests. In the same study, they reported that leukocyte, CRP, and LDH values were risk factors for intestinal necrosis as a result of the analysis and that the probability of intestinal necrosis increased 10 times with values of 7 and above in the Malatya Volvulus Gangrene Model (MVGM) they created. In addition, the enzymatic activity of LDH, which catalyzes the conversion of pyruvate to lactate, has been associated with intestinal ischemia in both experimental[17] and human[18] studies.

Contrary to the literature, in the analyzes performed on 47 patients in our study, no statistically significant difference was found between the groups with and without intestinal necrosis in WBC, Neutrophil count, Lymphocyte count, Monocyte count, Platelet count, RDW value, NLR, Lactate level, CRP, Amylase, and LDH values.

The important limitations of this study are the small number of cases included and the retrospective study.

CONCLUSION

Although inflammatory parameters were found to be significant in predicting bowel necrosis in the literature, it was concluded in this study that these parameters were not significant in predicting necrosis in patients with SV.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Ankara City Hospital No:1 Clinical Researches Ethics Committee (Date: 12.01.2022, Decision No: E1-22-2275).

Informed Consent: The written informed consents were obtained. All patients signed and free and informed consent form.

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